Exercise and oxygen debt. It should be noted that the subjects were healthy without heart disease. The increased postexercise oxygen debt was observed in subjects using a a bicycle ergometer which increases the oxygen consumption 20 to 25 fold:

(not cited) Chevalier et al. (1963) from 300 to 5,833 cc/min

(64) Krumholz et al. (1964) from 300 to 7,900 cc/min.

Such enormous increase in oxygen consumption is not encountered in ordinary work.

This paragraph implies that since carbon monoxide inhalation increases postexercise oxygen debt and smokers also show the effect, that carbon monoxide is the cause in smokers. Reference (21) Chevalier et al. specifically stated that the pathogenesis is unknown. Their summary is as follows:

> Differences have been found in the cardiopulmonary responses to exercise between young male cigarette smokers and nonsmokers. The present study was undertaken in an attempt to determine the cause of such differences. Since smokers carry a chronically elevated carboxyhemoglobin level (>4%), carbon monoxide was inhaled by nonsmokers to raise their carboxyhemoglobin level to the range seen in a control group of smokers. This maneuver caused the development in nonsmokers of an increased oxygen debt with exercise and a reduced pulmonary diffusing capacity at rest. The changes after carbon monoxide inhalation were similar to those found when comparing smokers to nonsmokers. The pathogenesis of these changes are unknown, but support carbon monoxide as a possible etiologic factor accounting for the less efficient exercise and poorer pulmonary function performance of smokers compared to nonsmokers.

. . . .

In the articles cited above, there are differences in responses to exercise of smokers and of nonsmokers inhaling carbon monoxide. The latter show slowing of the heart rate and decreased oxygen uptake during the working period; smokers show tachycardia and increased oxygen uptake.

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Source: https://www.industrydocuments.ucsf.edu/docs/xtlk0000